Van Ordt, feeding a number of patients suffering from various nervous diseases, with an excess of glycose and dextrose, found a large percentage (14 per cent.) presented glycosuria phenomena as a result. The experiment is hardly a fair one in view of the almost constant presence in neuropathic patients of gastric or intestinal disorder in some form. The experiment included 178 patients with widely different maladies, and the general conclusions to be drawn are not important. As might have been anticipated, glycosuria was not found constantly associated with any special diseases, but was a little more frequent with diseases of the encephalon. No doubt in many cases it was present as an expression of the general lowering of nutrition; but nevertheless it is true that its appearance and its fluctuations with the intensity of the nervous disease testify to a relation with pathological processes in the central nervous system.

In glycosuria consequent upon trauma the symptoms appear to be related rather to the nervous phenomena, to the traumatic neurosis, that is to say, than to the injury itself, and again individual predisposition must be taken into account, else we cannot explain why the same grave psychic syndromes should determine glycosuria in one case and not in another.

Glycosuria was noted in neurasthenia, hysteria and post-traumatic neurosis; it was not found in other neuroses, in true epilepsy, nor in general in spinal diseases not affecting the bulb.

W. Ebstein has examined the relations between epilepsy and diabetes from three points of view: 1, epilepsy as a result of diabetes; 2, diabetes as a result of epilepsy, and 3, the two maladies as a result of one cause.

The first is not a frequent case. In sixteen cases of diabetic coma Dreschfeld saw convulsions only once, and in eighty reported cases of coma but six cases of convulsions are noted, where albuminuria was not present to account for the seizures.

Jacoby considers diabetic epilepsy as an intermittent acetonæmic manifestation.

The second may be subdivided into two classes, according to whether there is simply a passing glycosuria after the attacks or a chronic diabetes. A passing glycosuria has been noticed by some former authors, but neither Ebstein nor Huppert has seen it, a result which agrees with Van Ordt.

True diabetes consequent upon epilepsy has never been noted except in Griesingers's case, which he attributed to an abuse of sugary food.

As to the third class, the causes may be general or local; hereditary predisposition and family habit may play a large part in both disorders, but the co-existence of the two diseases is certainly not common. Ebstein quotes one case. In two other cases with symptoms pointing to hemi-lateral cerebral lesions the patients suffered also with intermittent diabetes. Ebstein concluded that if glycosuria is not more frequently observed in epileptic patients, especially those suffering from the Jacksonian type of the disease, it is only because the urine is not frequently enough examined for sugar.

MITCHELL.

259. ZUR MULTIPLEN HERDSKLEROSE (Concerning Multiple Sclerosis). M. Probst (Deutsche Zeitschrift für Nervenheilkunde, 12, 1897-1898, p. 446).

The writer reports a case which presented clinically the appearance of amyotrophic lateral sclerosis, but proved on microscopical examination to be one of disseminated sclerosis. A man of fifty-four years had bulbar symptoms, with muscular atrophy and spastic paresis. Disturbance of deglutition and speech, atrophy of the tongue and lips, bilateral facial paralysis, atrophy of the small muscles of the hands, spastic paresis of the lower extremities, indicated involvement of the motor tracts. The classical symptoms of multiple sclerosis, intention tremor, scanning speech, ocular disturbance, etc., were not present, but the patient had vertigo and a somewhat slow reaction of the pupils. The lesions were confined to the medulla oblongata, pons and a portion of the corpora quadrigemina. Muscular atrophy is rarely seen in multiple sclerosis, but has been reported, and usually no change has been found in the ganglion cells of the anterior horns. Probst found in his case diminution in number and shrinkage of these cells in the upper part of the cervical cord. Secondary degeneration of the direct and crossed pyramidal tract was especially noteworthy, and extended from the proximal end of the pons into the lumbar cord. It was apparently due to a sclerotic area in the pons. Secondary degeneration has only been observed in a few cases of disseminated sclerosis. The columns of Goll were sclerotic from the upper thoracic region to the nuclei of these columns, and the intensity of the process was greatest in the upper part of the cord. The bulbar symptoms were explicable on account of the affection of the bulbar nuclei and nerves. Probst does not believe that this case was a combination of disseminated sclerosis and amyotrophic lateral sclerosis, because the vertigo was the first sign of the disease, because the muscular atrophy did not progress as rapidly as it usually does in amyotrophic lateral sclerosis, because the degeneration of the cells of the anterior horns was not as great as is usually seen in this affection, and because the secondary degeneration of the pyramidal tracts began in a sclerotic focus.

From a study of fifty-eight clinical cases of disseminated sclerosis, Probst finds that the sexes are about equally affected, that the disease is most common between twenty and thirty years of age, that exposure to cold and wet is the most common cause, and that infectious processes come next in etiological importance.

SPILLER.

260. EIN FALL VON ISOLIERTER FACIALIS-UND HYPOGLOSSUS-LAEHMUNG NEBST PSYCHISCHER ALTERATION INFOLGE VON TYPHUS ABDOMINALIS (A Case of Isolated Paralysis of the Facial and Hypoglossal Nerves with Psychical Alteration in Consequence of Typhoid Fever). A. Friedländer (Monatsschrift für Psychiatrie und Neurologie, 4, 1898, p. 110).

A boy, not hereditarily afflicted but not of normal psychical development, presented no unusual symptoms until he became fourteen or fifteen years old. He had been obedient until this time, but now became disobedient and excited, spoke much, ran from the house without any known motive, etc.; later he became more quiet and was able to work. He had a severe attack of typhoid fever, and during the convalescence he manifested symptoms of acute dementia and had paralysis of the right facial nerve. His behavior was that of a little child. He exhibited mind blindness, forced laughter and periodic attacks of intense anger, and made bad poetry; in short, he presented the symptom-complex of dementia præcox. The paralysis of the right facial nerve and, to some extent of the right hypoglossal, was not believed to be peripheral, as the electrical reactions were normal; or cortical, as sensation was not affected. It was thought to be the result of a small area of softening or of a hemorrhage in the white